

Case Report: Acquired Antisocial Personality Disorder Associated with Unilateral Left Orbital Frontal Lobe Damage

Christina A. Meyers¹, Stephen A. Berman¹, Randall S. Scheibel¹, Anne Hayman²

¹Department of Neuro-Oncology, The University of Texas, M.D. Anderson Cancer Center

²Department of Neuro-Radiology, Baylor College of Medicine

Accepted: June 10, 1992

We report on our analysis of a patient who developed personality changes which strongly resembled an antisocial personality disorder after surgical resection of a pituitary tumor. Despite behavioral changes that were obvious to friends, family and health care professionals, formal neuropsychological and personality testing revealed no specific cognitive deficits or psychopathology. We hypothesize that damage to a circumscribed region of the left orbitofrontal cortex, illustrated by magnetic resonance imaging, underlies these personality alterations. In contrast to previous reports, which ascribe such personality changes to bilateral frontal lobe injury, we suggest that unilateral frontal lobe damage alone may have resulted in the development of this syndrome.

Key Words: frontal lobe, personality disorder, neuropsychological testing

Recent neuropsychologic research has demonstrated that damage to frontal brain regions can produce recognizable psychologic symptom clusters. In particular, Eslinger and Damasio (1985) explored the mechanisms underlying antisocial behaviour in the context of specific damage to portions of the orbital and lower mesial frontal lobes. One notable feature of their study was documentation of a dissociation between the normal performance of their patient, E.V.R., on neuropsychological tests and his abnormal responses to real-life situations. E.V.R. had bilateral but relatively circumscribed frontal lobe damage involving Brodmann's cytoarchitectonic areas 11, 22, and 25 on the right and partial damage to areas 11 and 12 on the left, with subjacent loss of white matter. In addition, he sustained damage to mesial cortical areas 8 and 9 on the left and areas 32, 10, 8, 9, 24 and part of 6 on the right. Some dorsolateral cortex (areas 8, 9 and 46) on the right, and some white matter subjacent to the premotor cortex was also involved.

We had the opportunity to study an individual who exhibited personality changes similar to those of E.V.R., but who had a smaller unilateral lesion in the left orbitofrontal lobe.

The neuropsychiatric, neurological and radiographic analysis of our case allowed us to refine our hypotheses on the anatomic substrates of alterations in socially appropriate behavior.

CASE REPORT

J.Z. is a right-handed man who underwent surgical excision of a pituitary adenoma in 1962, at the age of 33. At that time, he had been working as a retail clothing salesman and experienced initial symptoms, which included progressive headaches, memory loss and fatigue. He also had a poor appetite and a two-year history of decreased libido. During his presurgery examination, J.Z. was confused and demonstrated bitemporal hemianopia, gynecomastia and galactorrhea.

A left frontal craniotomy was performed on March 30, 1962, and approximately 30% of a chromophobe adenoma was removed by a transfrontal approach. After surgery, the patient received radiation therapy; he also received hormone replacement therapy. His medical condition remained stable until 1986, when he developed nausea, vomiting and confusion. CT scan indicated obstructive hydrocephalus secondary to residual pituitary tumor. A ventriculoperitoneal shunt was placed through the left frontal lobe, and the patient's symptoms subsequently improved. An endocrinological evalua-

Address reprint requests to: Dr. Christina A. Meyers, Department of Neuro-Oncology (100), M.D. Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, Texas USA 77030.

Table 1
Neuropsychological test findings

Domain	Test	Subtest	Score	
Intellect	WAIS-R	Information	12 ^a	
		verbal IQ=105 (average)	Digit span	14
		performance IQ=98 (average)	Vocabulary	10
			Arithmetic	11
			Comprehension	8
			Similarities	10
			Picture completion	8
			Picture arrangement	9
			Block design	13
			Object assembly	11
			Digit symbol	9
			Memory	Wechsler Memory Scale
Logical memory – delayed	12			
Benton Visual Retention Test (Administration A: Form C)	Number correct	5		
	Number of errors	6		
Language	Multilingual Aphasia Examination	Visual naming	27 ^b	
		Word fluency	30	
		Token test	9	
Visual perception		Facial recognition	88 ^b	
		Road map test	18	
		Trails Part A	25	
		Trails Part B	40	
Executive functions	Wisconsin Card Sorting	Categories achieved	6	
		Perseverative errors	1	
		Total errors	10	
		Category Test	Total errors	15

^a age-corrected scaled scores; ^b percentiles

tion performed after shunt placement revealed a very high level of prolactin (930 ng/ml) produced by the residual tumor. The patient then began ongoing treatment with the dopamine agonist bromocriptine to control prolactin production and shrink the tumor.

Recent MRI scans revealed a stable, chronic enlargement of the sella turcica with residual tumor, most of which was on the left side. The mass extends superiorly and posteriorly into the interpeduncular cistern. Post-surgical encephalomalacia is also seen in the orbital left frontal lobe, including the gyrus rectus, olfactory sulcus and orbital gyri. The region in question corresponds to Brodmann's areas 12 and 11 (see Figs. 1 and 2).

The most notable aspects of this case are J.Z.'s obvious behavior changes after surgery. Before his symptoms began in 1962, J.Z. was reportedly an honest, stable and reliable worker and husband. He had worked at the same store for a number of years without incident. According to his wife and children, J.Z. manifested marked personality changes after surgical resection of the tumor in 1962. These personality changes included suspiciousness, irresponsible behavior at work and at home and grandiose beliefs about business deals, which the patient said would make him "millions, billions and trillions" of dollars. He was never able to return to regular employment after his surgery, and his wife divorced him because he lost much of their personal savings in several

questionable business deals. At the time of our assessment, he was unemployed and living with his mother. The brother who accompanied him to the assessment reported that J.Z. always said that he was on the verge of making “billions” of dollars in multinational business deals, but that his fiscal irresponsibility left his family with a number of debts.

The patient was referred to us for neuropsychologic evaluation in October 1987 for assessment of his personality and cognitive functioning. A summary of the test scores is presented in Table 1. J.Z. obtained a full-scale IQ score of 101 on the Wechsler Adult Intelligence Scale–Revised, a score in the average range (Wechsler 1981). The subtest scores ranged from low-average on tests of social judgement (comprehension) and identifying essential elements (picture completion) to superior on digit span. J.Z.’s borderline score on a test of language comprehension (Benton and Hamsher 1983) was caused by his impulsive responses (i.e., he responded before hearing the complete command). Performance on tests generally sensitive to frontal lobe executive functions — the Wisconsin Card Sorting Test (Heaton 1981) and the Booklet Category Test (DeFilippis and McCampbell 1979) — was high-average to superior. Personality assessment using the Minnesota Multiphasic Personality Inventory (MMPI) revealed a high degree of defensiveness, and he had a slightly elevated score on the scale measuring endorsement of multiple somatic complaints. There was no evidence of other psychopathology.

The patient’s behaviour during testing was the most notable aspect of the evaluation. J.Z. was impulsive, disinhibited, and tangential. His mood was labile, and he became agitated and upset discussing certain topics, then jovial and euphoric when the subject was changed. He freely reported being involved in criminal activities and said that he had three billion dollars hidden away in West Germany. However, he did not complain of hallucinations and was not psychotic. J.Z.’s disturbances of behavior and social judgement in the

context of intact cognitive functioning were consistent with the diagnosis of acquired antisocial personality disorder.

DISCUSSION

This case demonstrates the effects of unilateral, circumscribed orbitofrontal brain injury on personality and behavior. J.Z.’s post-surgical personality changes — disinhibition, poor judgement and irresponsibility toward his familial and social obligations — can be best explained by damage to the left orbitofrontal region resulting from surgery. Similar personality syndromes without marked cognitive dysfunction have occurred in other patients who had more extensive bilateral damage to the orbitofrontal regions (Eslinger and Damasio 1985; Vanderploeg and Haley 1990). Our case therefore supports the hypothesis that damage to the orbitofrontal cortex can cause this type of personality disorder and refines the notion of the minimal requisite substrate damage.

A number of diagnostic labels have been used in the literature to describe personality changes secondary to brain injury. The terms “sociopathic” and “psychopathic personality” were often used in early reports (Goodwin and Guze 1979). Acquired personality states which resembled these were often called “pseudosociopathic” or “pseudopsychopathic” (Benson and Blumer 1975). The currently preferred term, according to the DSM-III-R (American Psychiatric Association 1987), is “antisocial personality disorder.” This disorder is characterized by “a pattern of irresponsible and antisocial behavior beginning in childhood or early adolescence and continuing into adulthood.” As an adult, the patient must have at least four of the following characteristics: 1. lack of consistent work behavior; 2. non-conformance to usual social norms; 3. tendency to be irritable and aggressive; 4. repeated problems honoring financial obligations; 5. failure to plan ahead or impulsive behavior; 6. untruthfulness; 7. recklessness with regard to personal safety;

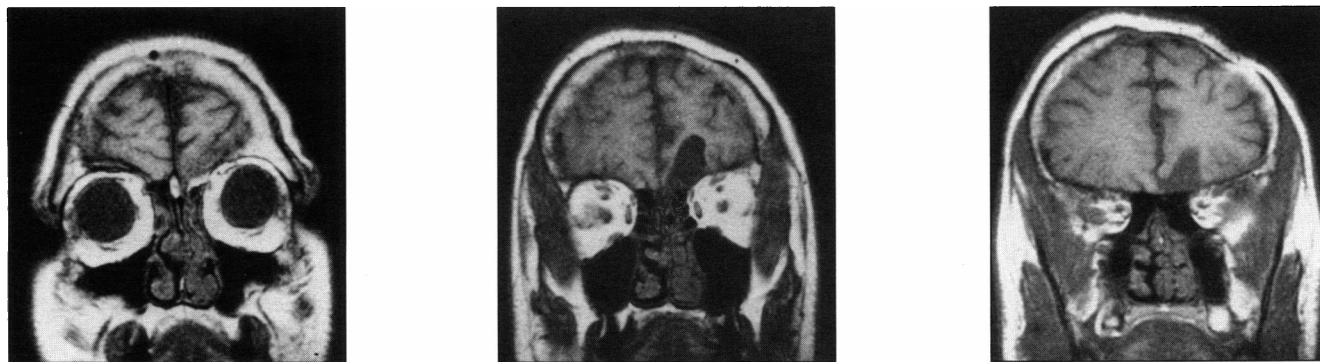


Fig. 1: Coronal MRI scans show a wedge-shaped zone of cerebral damage in the left orbitofrontal lobe involving the gyrus rectus and the medial aspect of the orbital gyri. Note that part of the medial cortex of the gyrus rectus is intact.

8. inability to function as a responsible parent; 9. inability to sustain a monogamous relationship for more than one year; and 10. lack of remorse. Our analysis of J.Z.'s behavior indicated that he exhibited at least characteristics 1, 4, 5, 6 and 10 of the above. He also met the further criterion of being over the age of 18. However, he did not meet the criterion of having a conduct disorder which began in childhood. We have characterized J.Z.'s post-surgical changes in behavior as consistent with an acquired antisocial personality disorder.

The literature describing the effects of frontal lobe injury on the regulation of affect and behavior is difficult to interpret since different patient populations have been studied. Individuals with no history of neurological impairment who have been diagnosed with sociopathic or antisocial personality disorders do not appear to have associated frontal lobe dysfunction (Kandel and Freed 1989). Patients with diffuse head injuries with additional focal lesions of the orbitofrontal lobes tend to have poor insight, disinhibition and poor planning, in addition to cognitive deficits. These symptoms have been described as defects in metacognition and are similar to the personality changes seen in J.Z. (Mattson and Levin 1990). In a thorough study of the effects of unilateral frontal lesions on mood and behavior, Grafman and colleagues

(1986) found that patients with left dorsolateral and right orbitofrontal lesions had the most pronounced disorders. In that study, persons with left dorsolateral damage tended to display more anger and hostility, whereas those with right orbitofrontal lesions were more anxious. Patients with left orbitofrontal lesions, as in the case of J.Z., tended to be highly defensive and to present themselves in "an angelic light." However, this study used self-reports to assess mood and did not address the ability of these individuals to function appropriately in daily life, i.e., how their reported symptomatology was expressed in their environment. The objective observations in Grafman's study were limited to behavior resembling anxiety.

In general, two categories of personality change have been associated with frontal lobe disease. One group tends to display "apathy and indifference" and the other tends to exhibit "puerility and euphoria" (Benson and Blumer 1975). Although combinations of these characteristics occur, the first type is usually related to damage to the frontal convexities and associated subcortical regions. The second personality type occurs with lesions to the orbital surface, including the mesial frontal areas (Eslinger and Damasio 1985). Prior to the analysis of E.V.R., it had generally been assumed that

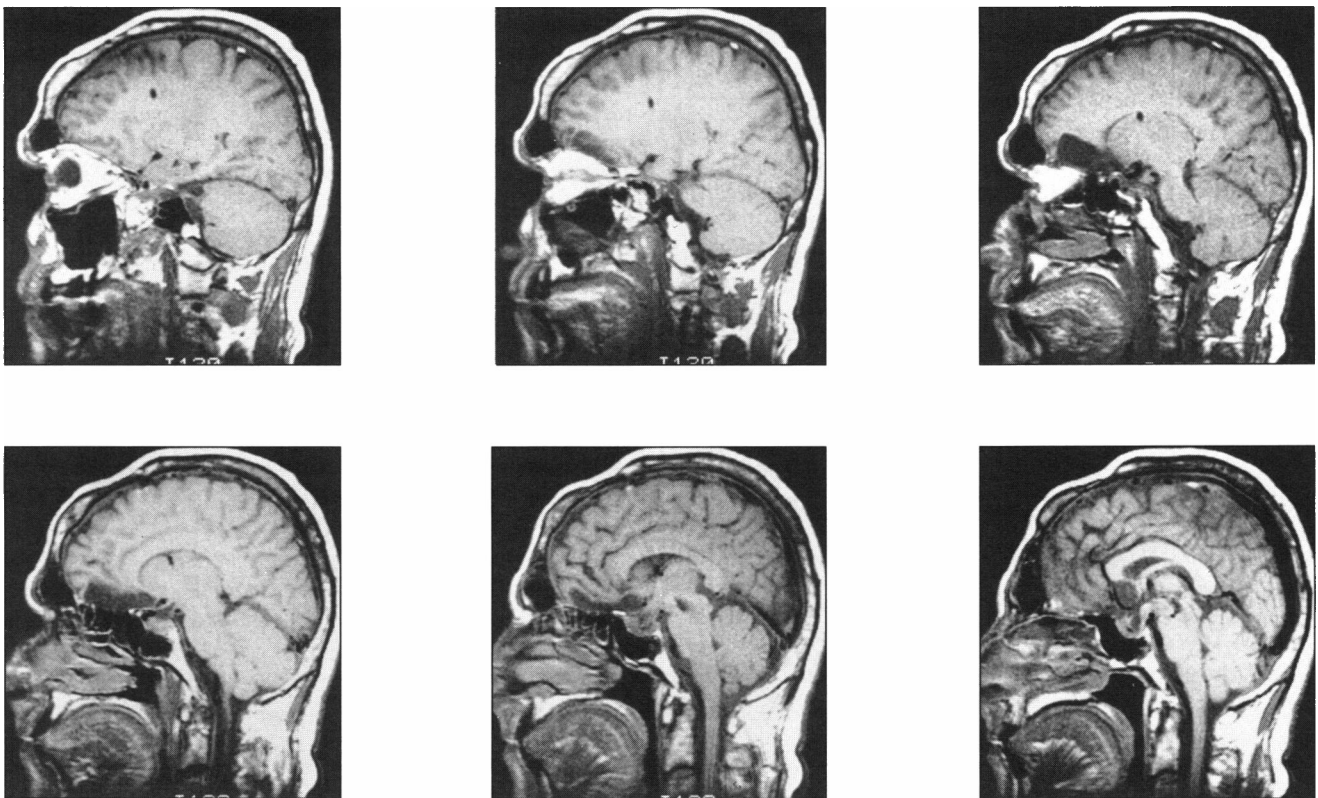


Fig. 2: Sagittal MRI scans show the zone of cerebral damage described in Fig. 1. Note sparing of the frontal cortex anterior to this zone and the post-operative abnormality in the sellar region.

individuals displaying either type of frontal lobe personality change would also have cognitive deficits which would be detectable by psychological tests sensitive to frontal lobe dysfunction. However, the case of E.V.R. demonstrated a dissociation between normal performance on such tests and impairment of behavior in the course of carrying out activities of daily living. The relatively limited area of bilateral frontal damage, restricted mainly to the orbital areas, appeared to be the key to this dissociation (Eslinger and Damasio 1985). The authors hypothesized that E.V.R. had difficulty analyzing and integrating real-life situations in the context of the behavioral knowledge which he still possessed and that he had difficulty executing the results of such analysis and integration. Eslinger and Damasio speculated that E.V.R.'s problems were based on the loss of interaction between the damaged cortical areas and the dorsolateral cortex and limbic system. Similar cases have involved somewhat more extensive bilateral lesions (Vanderploeg and Haley 1990), and thus the minimal lesion necessary for the production of this syndrome had not been further refined. In particular, the commonly accepted notion that bilateral damage is necessary (Benson and Blumer 1975) has yet to be seriously challenged.

Although J.Z. has had a complicated medical history, it is unlikely that factors other than left orbitofrontal lobe damage are responsible for the changes in his behavior. Brain irradiation may cause delayed white matter degeneration, but the MRI scans, which are sensitive indicators of white matter changes, show no such degeneration (Curnes et al 1986). Although hyperprolactinemia in itself has been associated with changes in mood, these have been described as difficulties with depression and anxiety (Thienhaus and Hartford 1986; Fava et al 1982). Finally, J.Z.'s symptomatology is unlikely to be a side-effect of bromocriptine therapy (Serby et al 1978; Einarson and Turchet 1983); his personality changes occurred long before bromocriptine therapy was initiated and continued unabated through a four-week trial discontinuation of bromocriptine. However, we cannot entirely eliminate the possibility that the primary pituitary pathology or subsequent alterations in the hypothalamic-pituitary axis contributed to the observed psychopathology.

In summary, this case suggests that injury restricted to the left orbitofrontal lobe may be sufficient to produce a personality disorder resembling an antisocial personality. This finding raises several intriguing questions. Is there greater lateralized processing specificity in the frontal regions than has been reported, or can this syndrome result from injury to the homologous right orbitofrontal lobe? The role of the disruption of frontolimbic and frontoparietal connections also deserves further study with regard to the pathogenesis of such personality disorders.

REFERENCES

- American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised*. Washington, DC: American Psychiatric Press, Inc.
- Benson DF, Blumer D (1975) *Psychiatric Aspects of Neurologic Diseases*. New York: Grune and Stratton, pp 152-158.
- Benton AL, Hamsher KdeS (1983) *Multilingual Aphasia Examination*. Iowa City, IA: AJA Associates, Inc.
- Curnes JT, Laster DW, Ball MR, Moody DM, Witcofski RL (1986) MRI of radiation injury to the brain. *American Journal of Radiology* 147:119-124.
- DeFilippis NA, McCampbell E (1979) *The Booklet Category Test*. Odessa, FL: Psychological Assessment Resources, Inc.
- Einarson TR, Turchet EN (1983) Psychotic reaction to low-dose bromocriptine. *Clin Pharmacol* 2:273-274.
- Eslinger PJ, Damasio AR (1985) Severe disturbance of higher cognition after bilateral frontal lobe ablation: patient EVR. *Neurology* 35:1731-1741.
- Fava M, Fava GA, Kellner R, Serafini E, Mastrogiacomo I (1982) Psychological correlates of hyperprolactinemia in males. *Psychother Psychosom* 37:214-217.
- Goodwin DW, Guze SB (1979) *Psychiatric Diagnosis, Second Edition*. New York: Oxford University Press.
- Grafman J, Vance SC, Weingartner H, Salazar AM, Amin D (1986) The effects of lateralized frontal lesions on mood regulation. *Brain* 109:1127-1148.
- Hathaway SR, McKinley JC (1970) *Minnesota Multiphasic Personality Inventory*. Minneapolis, MN: National Computer Systems.
- Heaton RK (1981) *The Wisconsin Card Sorting Test*. Odessa, FL: Psychological Assessment Resources, Inc.
- Kandel E, Freed D (1989) Frontal-lobe dysfunction and antisocial behavior: a review. *J Clin Psychol* 45:404-413.
- Mattson AJ, Levin HS (1990) Frontal lobe dysfunction following closed head injury: a review of the literature. *J Nerv Ment Dis* 178:282-291.
- Serby M, Angrist B, Lieberman A (1978) Mental disturbances during bromocriptine and lergotril treatment of Parkinson's disease. *Am J Psychiatry* 135:1227-1229.
- Thienhaus OJ, Hartford JT (1986) Depression in hyperprolactinemia. *Psychosomatics* 27:663-664.
- Vanderploeg RD, Haley JA (1990) Pseudosociopathy with intact higher-order cognitive abilities in patients with orbitofrontal cortical damage. *J Clin Exp Neuropsychol* 12:54-55.
- Wechsler D (1981) *Wechsler Adult Intelligence Scale-Revised*. San Antonio, CA: The Psychological Corporation.